7/99 POD

PAT' ENTERED AT 09:11:32 ON 22 JUL 1999)
L1 0 S EHEC/CLM AND EPEC/CLM
L2 3 S ENTEROPATHOGEN?/CLM AND (EHEC/CLM OR ENTEROHEMORRHAG?)
L3 1 S ENTEROPATHOGEN?/CLM AND (EHEC/CLM OR ENTEROHEMORRHAG?/CL
M)
L4 2 S L2 NOT L3
L5 1 S ENTERHEMORRHAG?/CLM
L6 0 S EHEC/TI AND EPEC/TI
L7 229 S EHEC
L8 24 S EPEC
L9 9 S L7 AND L8
L10 9 S L7 (P) L8
L11 5 S L10 AND (POLYCLONAL? OR MONOCLONAL? OR ANTISER? OR ANTIB
OD?
L12 1 S L11 AND EAEA
L13 0 S EAEA/TI
L14 1 S INTIMIN?/TI
L15 12 S EAEA
L16 0 S ENTERVIRUL?/TI
L17 5 S ENTEROVIR?/TI
L18 0 S ENTEROVIRUL?/TI
L19 0 S VEROTOX?/TI
L20 5 S ENTEROHEMORR?/TI
L21 4 S ENTEROPATHO?/TI
L22 0 S L20 AND L21
L23 9 S L20 OR L21
L24 6 S L23 NOT L10
L25 2 S O157?/TI
L26 8 S 0157:H7/TI
L27 3 S L26 NOT (L23 OR L10)
L28 2 S ANTIBOD?/TI AND L7
L29 0 S IMMUNOGLOB?/TI AND L7
L30 0 S ANTISER?/TI AND L7
L31 0 S MONOCLONAL?/TI AND L7
L32 2 S (ANTIBOD? OR IMMUNOGLOB? OR MONOCLONAL? OR MONOSPECIFIC?
O

09850378 BIOSIS NO.: 199598305296
Co-expression of the B subunit of %shiga%-like toxin I and %EaeA% from enterohemorrhagic Escherichia coli in Vibrio cholerae vaccine strains.

AUTHOR: Butterton Joan R; Ryan Edward T; Calderwood Stephen B
AUTHOR ADDRESS: Massachusetts General Hosp., Boston, MA, USA

JOURNAL: Abstracts of the General Meeting of the American Society for Microbiology 95 (0):p294 1995

CONFERENCE/MEETING: 95th General Meeting of the American Society for Microbiology Washington, D.C., USA May 21-25, 1995
ISSN: 1060-2011
RECORD TYPE: Citation
LANGUAGE: English

DESCRIPTORS:
MAJOR CONCEPTS: Cardiovascular System (Transport and Circulation); Digestive System (Ingestion and Assimilation); Genetics; Immune System (Chemical Coordination and Homeostasis); Infection; Molecular Genetics (Biochemistry and Molecular Biophysics); Pathology; Pharmacology
BIOSYSTEMATIC NAMES: Enterobacteriaceae--Eubacteria, Bacteria; Leporidae --Lagomorpha, Mammalia, Vertebrata, Chordata, Animalia; Vibrionaceae-- Eubacteria, Bacteria
ORGANISMS: rabbit (Leporidae); Escherichia coli (Enterobacteriaceae); Vibrio cholerae (Vibrionaceae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; bacteria; chordates; eubacteria; lagomorphs; mammals; microorganisms; nonhuman mammals; nonhuman vertebrates; vertebrates
MISCELLANEOUS TERMS: ANTIBODY RESPONSE; GENETICS; IMMUNOGLOBULIN A; IMMUNOGLOBULIN G; MEETING ABSTRACT; TRANSCRIPTIONAL REGULATION
CONCEPT CODES:
10300 Replication, Transcription, Translation
12508 Pathology, General and Miscellaneous-Inflammation and

07620438 93378160

Association between the effacing (%eae%) gene and the %Shiga%-like toxin-encoding genes in Escherichia coli isolates from cattle.

Mainil JG; Jacquemin ER; Kaeckenbeeck AE; Pohl PH

Department of Bacteriology, Faculty of Veterinary Medecine, University of Liege, Sart-Tilman, Belgium.

Am J Vet Res (UNITED STATES) Jul 1993; 54 (7) p1064-8, ISSN 0002-9645 Journal Code: 40C

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9312

Subfile: INDEX MEDICUS

Two hundred ninety-six Escherichia coli isolates from feces or intestines of calves with diarrhea were hybridized with 7 gene probes. One probe (the eae probe) was derived from the eae gene coding for a protein involved in the effacement of the enterocyte microvilli by the group of bacteria called attaching and effacing E coli (AEEC), and 2 probes were derived from genes coding for the Shiga-like toxins (SLT) 1 and 2 produced by the verocytotoxic E coli (VTEC). The other 4 probes were derived from DNA sequences associated with the adhesive properties of enteroadherent E coli (EAEC) to cultured cells (the EAF probe for the localized adherence pattern, probes F1845 and AIDA-1 for the diffuse adherence pattern, and the Agg probe for the aggregative adherence pattern). Hybridization results for the eae probe were in agreement, for all but 1 of the 8 isolates, with previously published phenotypic results of microvilli effacement. The latter was previously reported as effacing the microvilli of calf enterocytes, but was eae probe-negative. Two classes of isolates hybridized with the eae probe. Members of a first class (60 isolates) additionally produced a positive signal with 1 or both of the SLT probes (VTEC-AEEC isolates). Isolates hybridizing with the eae and the SLT1 probes were the most frequent: 56 isolates (ie, 93% of all VTEC-AEEC). Members of the second class (10 isolates) failed to hybridize with either SLT probe (non-VTEC-AEEC isolates).(ABSTRACT TRUNCATED AT 250 WORDS)

Tags: Animal

Descriptors: *Bacterial Toxins--Genetics--GE; *Cattle--Microbiology--MI; *Enterotoxins--Genetics--GE; *Escherichia coli--Genetics--GE; *Genes, Bacterial; DNA Probes; DNA, Neoplasm--Genetics--GE; Escherichia coli --Classification--CL; Escherichia coli--Isolation and Purification--IP; Plasmids; Restriction Mapping; Serotyping

CAS Registry No.: 0 (Bacterial Toxins); 0 (DNA Probes); 0 (DNA, Neoplasm); 0 (Enterotoxins); 0 (Plasmids); 0 (Shiga-like toxin I); 0 (Shiga-like toxin II)

Gene Symbol: eae; Agg

S (EAE OR EAEA OR INTIMIN)(50N) (EAEB)(50N) (ANTISER? OR ANTIBOD? OR M-ONOCLONAL? OR POLYCLONAL?)

Ref	Items	File
N1	5	155: MEDLINE(R)_1966-1999/Sep W2
N2	1	151: HealthSTAR_1975-1999/Aug
N3	1	156: Toxline(R)_1965-1999/Jun
N4	0	2: INSPEC_1969-1999/Jul W2
N5	0	5: Biosis Previews(R)_1969-1999/Jun W4
N6	0	6: NTIS_64-1999/Aug W3
N7	0	8: Ei Compendex(R)_1970-1999/Jul W2
N8	0	9: Business & Industry(R) Jul_1994-1999/Jul 22
N9	0	10: AGRICOLA_70-1999/Jun
N10	0	14: Mechanical Engineering Abs_1973-1999/Jul

3 files have one or more items; file list includes 254 files.

- Enter P or PAGE for more -

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            FTSNET   0.116 Hrs.
     $7.10  Estimated cost this search
     $7.86  Estimated total session cost   5.932 DialUnits


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  File 151:HealthSTAR  1975-1999/Aug
        (c) format only 1999 The Dialog Corporation
*File 151: Reloaded. Note accession numbers changed.
  File 156:Toxline(R)  1965-1999/Jun
        (c) format only 1999 The Dialog Corporation

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             157  EAEA
             113  INTIMIN
              54  EAEB
           58489  ANTISER?
          678087  ANTIBOD?
          176313  MONOCLONAL?
           35056  POLYCLONAL?
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                  ANTIBOD? OR MONOCLONAL? OR POLYCLONAL?)
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...completed examining records
      S2       5  RD (unique items)
?t s2/9/all

 2/9/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 1999 Dialog Corporation. All rts. reserv.

09816456   99059882
 Antibody response of children with enteropathogenic Escherichia coli
infection to the bundle-forming pilus and locus of enterocyte
effacement-encoded virulence determinants.
  Martinez MB; Taddei CR; Ruiz-Tagle A; Trabulsi LR; Giron JA
  Faculdade de Ciencias Farmaceuticas, Universidade de Sao Paulo, Brazil.
mbmartin@usp.br
  J Infect Dis (UNITED STATES) Jan 1999, 179 (1) p269-74, ISSN
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0022-1899 Journal Code: IH3
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9904
Subfile: AIM; INDEX MEDICUS

Enteropathogenic Escherichia coli (EPEC) express a plasmid-encoded type IV pilus termed bundle-forming pilus, which is associated with the formation of bacterial microcolonies on cultured epithelial cells. Bacterial attachment and effacement of the enterocyte brush border membrane is attributed to a surface outer membrane protein adhesin termed intimin and EPEC-secreted proteins EspA, EspB, and EspD. Except for intimin, production in vivo or antibody response against these virulence determinants during natural EPEC infections in young children has not been demonstrated. Antibody responses against BfpA, intimin, EspA, and EspB were investigated in Brazilian children naturally infected with EPEC. Generally, IgG antibodies against BfpA and EspB were the most commonly found, followed by anti-EspA and intimin antibodies. Thus, bundle-forming pilus and locus of enterocyte attachment-encoded products are produced in vivo during natural EPEC infections and elicit an immune response against heterologous EPEC virulence determinants. These findings have important implications in the immunoprophylaxis against EPEC infections.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: *Antibodies, Bacterial--Biosynthesis--BI; *Escherichia coli --Immunology--IM; *Escherichia coli--Pathogenicity--PY; *Escherichia coli Infections--Immunology--IM; *Fimbriae, Bacterial--Immunology--IM; Antigens, Bacterial--Genetics--GE; Bacterial Adhesion--Genetics--GE; Bacterial Adhesion--Immunology--IM; Bacterial Outer Membrane Proteins--Genetics--GE; Bacterial Outer Membrane Proteins--Immunology--IM; Bacterial Proteins --Genetics--GE; Bacterial Proteins--Immunology--IM; Case-Control Studies; Child, Preschool; Diarrhea--Immunology--IM; Diarrhea--Microbiology--MI; Diarrhea--Prevention and Control--PC; Epithelial Cells--Microbiology--MI; Escherichia coli--Genetics--GE; Escherichia coli Infections--Microbiology --MI; Escherichia coli Infections--Prevention and Control--PC; Fimbriae, Bacterial--Genetics--GE; IgG--Biosynthesis--BI; Infant; Intestines --Microbiology--MI; Virulence--Genetics--GE; Virulence--Immunology--IM

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (BfpA protein); 0 (EaeB protein); 0 (EspA protein); 0 (IgG); 147094-99-3 (eae protein)

2/9/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09723304 98368029

Human colostrum contains IgA antibodies reactive to enteropathogenic Escherichia coli virulence-associated proteins: intimin, BfpA, EspA, and EspB.

Loureiro I; Frankel G; Adu-Bobie J; Dougan G; Trabulsi LR; Carneiro-Sampaio MM

Department of Immunology, University of Sao Paulo, Brazil.

J Pediatr Gastroenterol Nutr (UNITED STATES) Aug 1998, 27 (2) p166-71, ISSN 0277-2116 Journal Code: JL6

Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9901
Subfile: INDEX MEDICUS

BACKGROUND: In Brazil, enteropathogenic Escherichia coli diarrhoea is endemic among infants born into low economic levels, and it is one of the main causes of morbidity and mortality in this group. Binding of enteropathogenic E. coli to the brush border mucosa triggers a cascade of transmembrane and intracellular signals, causing cytoskeletal reorganization and formation of a specific lesion, termed the attaching and effacing lesion. Several enteropathogenic E. coli gene products have been implicated in formation of attaching and effacing lesions. Evaluation of pathogen-specific protective factors shows that breast feeding is effective

against enteropathogenic E. coli infection. To investigate the nature of the protection, defatted colostrum and secretory immunoglobulin A obtained from mothers living in Sao Paulo were investigated for the ability to recognise selected enteropathogenic E. coli-associated virulence factors. METHODS: Western blot analysis was used to investigate the IgA repertoire in pooled colostrum that is reactive with specific enteropathogenic E. coli proteins. Whole enteropathogenic E. coli bacterial cell extracts, nonpathogenic E. coli strains overexpressing specific virulence factors, and purified polypeptides were used as antigen sources in this study. RESULTS: Reaction of the colostrum samples in Western blots of whole bacterial cell extracts and selected purified enteropathogenic E. coli proteins showed that they contained a secretory immunoglobulin A reactive with all the virulence-associated proteins studied. CONCLUSION: These results suggest that maternal antibodies may protect infants from enteropathogenic E. coli infection by interfering with adherence processes (anti-intimin and anti-bundle-forming pili antibodies) and cell signaling (anti-enteropathogenic Escherichia coli-secreted protein A and B antibodies.

Tags: Female; Human; Support, Non-U.S. Gov't

Descriptors: *Antibodies, Bacterial--Analysis--AN; *Bacterial Proteins --Immunology--IM; *Colostrum--Immunology--IM; *Escherichia coli--Immunology --IM; *IgA--Analysis--AN; Adolescence; Adult; Antibodies, Bacterial --Immunology--IM; Bacterial Outer Membrane Proteins--Immunology--IM; Blotting, Western; Brazil; IgA--Immunology--IM; Signal Transduction

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (BfpA protein); 0 (EaeB protein); 0 (EspA protein); 0 (IgA); 147094-99-3 (eae protein)

2/9/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

09568571 98254135

Protein translocation into host epithelial cells by infecting enteropathogenic Escherichia coli.

Wolff C; Nisan I; Hanski E; Frankel G; Rosenshine I

Department of Molecular Genetics and Biotechnology, The Hebrew University, Faculty of Medicine, Jerusalem, Israel.

Mol Microbiol (ENGLAND) Apr 1998, 28 (1) p143-55, ISSN 0950-382X

Journal Code: MOM

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9809

Subfile: INDEX MEDICUS

Enteropathogenic Escherichia coli (EPEC) causes diarrhoea in young children. EPEC induces the formation of actin pedestal in infected epithelial cells. A type III protein secretion system and several proteins that are secreted by this system, including EspB, are involved in inducing the formation of the actin pedestals. We have demonstrated that contact of EPEC with HeLa cells is associated with the induction of production and secretion of EspB. Shortly after infection, EPEC initiates translocation of EspB, and EspB fused to the CyaA reporter protein (EspB-CyaA), into the host cell. The translocated EspB was distributed between the membrane and the cytoplasm of the host cell. Translocation was strongly promoted by attachment of EPEC to the host cell, and both attachment factors of EPEC, intimin and the bundle-forming pili, were needed for full translocation efficiency. Translocation and secretion of EspB and EspB-CyaA were abolished in mutants deficient in components of the type III protein secretion system, including sepA and sepB mutants. EspB-CyaA was secreted but not translocated by an espB mutant. These results indicate that EspB is both translocated and required for protein translocation by EPEC.

Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.

Descriptors: *Bacterial Outer Membrane Proteins--Metabolism--ME; *Epithelial Cells--Microbiology--MI; *Escherichia coli--Pathogenicity--PY; Antibodies, Bacterial--Immunology--IM; Bacterial Adhesion--Genetics--GE; Bacterial Outer Membrane Proteins--Genetics--GE; Bacterial Proteins

--Metabolism--ME; Cell Fractionation; Cell Membrane--Metabolism--ME; Cyclic AMP--Analysis--AN; Cyclic AMP--Metabolism--ME; Cytoplasm--Metabolism--ME; Epithelial Cells--Metabolism--ME; Escherichia coli--Genetics--GE; Escherichia coli--Metabolism--ME; Fimbriae, Bacterial--Physiology--PH; Genes, Bacterial; Hela Cells; Immunoblotting; Luminescent Proteins; Microscopy, Confocal; Protein Precursors--Metabolism--ME; Protein Processing, Post-Translational; Recombinant Fusion Proteins--Metabolism--ME ; Recombination, Genetic
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (EaeB protein); 0 (Luminescent Proteins); 0 (Protein Precursors); 0 (Recombinant Fusion Proteins); 121889-91-6 (cyclolysin); 147094-99-3 (eae protein); 147336-22-9 (green fluorescent protein); 60-92-4 (Cyclic AMP)

2/9/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09319225 98050926
Enteropathogenic E. coli (EPEC) transfers its receptor for intimate adherence into mammalian cells.
Kenny B; DeVinney R; Stein M; Reinscheid DJ; Frey EA; Finlay BB
Department of Biochemistry and Molecular Biology, University of British Columbia, Vancouver, Canada.
Cell (UNITED STATES) Nov 14 1997, 91 (4) p511-20, ISSN 0092-8674
Journal Code: CQ4
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9802
Subfile: INDEX MEDICUS
Enteropathogenic E. coli (EPEC) belongs to a group of bacterial pathogens that induce epithelial cell actin rearrangements resulting in pedestal formation beneath adherent bacteria. This requires the secretion of specific virulence proteins needed for signal transduction and intimate adherence. EPEC interaction induces tyrosine phosphorylation of a protein in the host membrane, Hp90, which is the receptor for the EPEC outer membrane protein, intimin. Hp90-intimin interaction is essential for intimate attachment and pedestal formation. Here, we demonstrate that Hp90 is actually a bacterial protein (Tir). Thus, this bacterial pathogen inserts its own receptor into mammalian cell surfaces, to which it then adheres to trigger additional host signaling events and actin nucleation. It is also tyrosine-phosphorylated upon transfer into the host cell.
Tags: Human; Support, Non-U.S. Gov't
Descriptors: *Bacterial Adhesion--Genetics--GE; *Bacterial Outer Membrane Proteins--Metabolism--ME; *Bacterial Proteins--Metabolism--ME; *Escherichia coli--Pathogenicity--PY; *Receptors, Cell Surface--Metabolism--ME; Amino Acid Sequence; Antibodies, Bacterial; Bacterial Outer Membrane Proteins --Genetics--GE; Bacterial Outer Membrane Proteins--Physiology--PH; Bacterial Proteins--Chemistry--CH; Bacterial Proteins--Genetics--GE; Bacterial Proteins--Isolation and Purification--IP; Bacterial Proteins --Physiology--PH; Base Sequence; Cell Membrane--Chemistry--CH; Cell Membrane--Metabolism--ME; Escherichia coli--Genetics--GE; Escherichia coli --Immunology--IM; Genes, Structural, Bacterial--Genetics--GE; Hela Cells; Isoelectric Point; Molecular Sequence Data; Molecular Weight; Mutation; Phosphorylation; Receptors, Cell Surface--Chemistry--CH; Receptors, Cell Surface--Genetics--GE; Receptors, Cell Surface--Isolation and Purification --IP; Recombinant Fusion Proteins--Analysis--AN; Restriction Mapping; Tyrosine--Metabolism--ME; Virulence
Molecular Sequence Databank No.: GENBANK/AF013122
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (EaeB protein); 0 (EspA protein); 0 (Receptors, Cell Surface); 0 (Recombinant Fusion Proteins); 0 (Tir protein); 147094-99-3 (eae protein); 55520-40-6 (Tyrosine)

2/9/5 (Item 5 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

08880014 97101056

Intimin from enteropathogenic Escherichia coli restores murine virulence to a Citrobacter rodentium eaeA mutant: induction of an immunoglobulin A response to intimin and EspB.

Frankel G; Phillips AD; Novakova M; Field H; Candy DC; Schauer DB; Douce G; Dougan G

Department of Biochemistry, Imperial College of Science, Technology and Medicine, London, United Kingdom. g.frankel@ic.ac.uk

Infect Immun (UNITED STATES) Dec 1996, 64 (12) p5315-25, ISSN 0019-9567 Journal Code: GO7

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9703

Subfile: INDEX MEDICUS

The formation of attaching and effacing (A/E) lesions is central to the pathogenesis of enteropathogenic Escherichia coli (EPEC)-mediated disease in humans and Citrobacter rodentium (formerly C. freundii biotype 4280)-mediated transmissible colonic hyperplasia in mice. Closely related outer membrane proteins, known as intimins, are required for formation of the A/E lesion by both EPEC (Int(EPEC)) and C. rodentium (Int(CR)). A secreted protein, EspB (formally EaeB), is also necessary for A/E-lesion formation. Here we report that expression of a cloned Int(EPEC), encoded by plasmid pCVD438, restores murine virulence to an intimin-deficient mutant of C. rodentium DBS255. Replacement of Cys937 with Ala abolished the ability of the cloned EPEC intimin to complement the deletion mutation in DBS255. Ultrastructural examination of tissues from wild-type C. rodentium and DBS255(pCVD438)-infected mice revealed multiple A/E lesion on infected cells and loss of contact between enterocytes and basement membrane. Histological investigation showed that although both wild-type C. rodentium and DBS255(pCVD438) colonized the descending colon and induced colonic hyperplasia in orally infected 21-day-old mice, the latter strain adhered to epithelial cells located deeper within crypts. Nonetheless, infection with the wild-type strain was consistently more virulent, as indicated by a higher mortality rate. All the surviving mice, challenged with either wild-type C. rodentium or DBS255(pCVD438), developed a mucosal immunoglobulin A response to intimin and EspB. These results show that C. rodentium infection provides a relevant, simple, and economic model to investigate the role of EPEC proteins in the formation of A/E lesions in vivo and in intestinal disease.

Tags: Animal; Human; Support, Non-U.S. Gov't

Descriptors: *Bacterial Outer Membrane Proteins--Toxicity--TO; *Citrobacter--Pathogenicity--PY; *Colon--Microbiology--MI; *Escherichia coli--Metabolism--ME; Antibodies, Bacterial--Biosynthesis--BI; Bacterial Outer Membrane Proteins--Genetics--GE; Bacterial Outer Membrane Proteins --Immunology--IM; Citrobacter--Genetics--GE; Citrobacter--Immunology--IM; Colon--Pathology--PA; Hyperplasia; IgA--Biosynthesis--BI; Mice; Mutation

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (EaeB protein); 0 (IgA); 147094-99-3 (eae protein)

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$0.36 Estimated cost File151

$0.30 0.118 DialUnits File156

$0.30 Estimated cost File156

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FTSNET 0.016 Hrs.

$3.28 Estimated cost this search

$11.14 Estimated total session cost 6.720 DialUnits

Your SELECT statement is:
s (SLT? OR SHIGA?)/TI AND (EAE OR EAEA OR EAEB)/TI

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4	10: AGRICOLA_70-1999/Jun
7	34: SciSearch(R) Cited Ref Sci_1990-1999/Jul W3
4	50: CAB Abstracts_1972-1999/Jun
5	51: Food Sci.&Tech.Abs_1969-1999/May
2	53: FOODLINE(R): Food Science & Technology_1972-1999/Jun 21
3	71: ELSEVIER BIOBASE_1994-1999/Jun W2
5	73: EMBASE_1974-1999/Jul W2
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1	151: HealthSTAR_1975-1999/Aug
7	155: MEDLINE(R)_1966-1999/Sep W2
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Examined 200 files	
Examined 250 files	

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